a) Filling in the Details

When the clinician has chosen a working hypothesis, he is faced with the problem of confirming the details of that hypothesis. Major research question are:

* How does he select the details to explore?
* What facts should he seek from the patient?
* How should he try to establish the facts he desires?
* In what sequence should he seek these facts?
* How does he assess the validity of information?

b) Assessing the 'goodness' of Fit

The clinician faces another problem when a more detailed piece of information concerning the patient has been obtained, regardless of the means. He must assess how well the new information 'fits' the current context. Further this assessment must be merged with similar assessments of the 'goodness of fit' of other facts. In the face of poorly fitting facts, how far should he pursue the current context before abandoning it?

One aspect of the assessment of the goodness of fit for a finding which is particularly interesting is the process by which alternative explanations are constructed for facts which appear to be discrepant with a given hypothesis. In such cases, the poor fit of a fact to a hypothesis does not immediately cause the rejection of a hypothesis, but rather it triggers a search for a way to 'explain away' the problem. In a later section, we will discuss in more detail the problem of discrepant information.

c) Rejecting Contexts

Above we mentioned that under certain circumstances, a context which was chosen by the clinician may be discarded by him, because of a 'poor fit' with the facts. In this case, the clinician is giving up the working hypothesis despite his initial desire to confirm it. Here, however, the principle of parsimony may make him reluctant to give up a particular hypothesis. For example, in abandoning the current hypothesis, he may be forced to hypothesize more than one disease. Although he is often forced to do this, the clinician, in general, is reluctant to do so, and so he may continue with a hypothesis which fits the facts rather poorly for longer than would otherwise be expected.

In other circumstances, however, the clinician may actively want to reject contexts. The most obvious example of this occurs when the clinician has found the working hypothesis to be a good fit to the presenting facts, and he now wants to reject any other competing hypotheses.

In many cases, the clinician remembers a specific pattern of the presence or absence of various signs and symptoms which virtually precludes the presence of a particular disease. In other cases, no such specific pattern is known to the clinician, and he must use other arguments (such as the relative likelihood of two hypotheses) to exclude the hypothesis in question. Of course, in certain cases, no such exclusion can be achieved, and he must base subsequent decisions on consideration of more than one hypothesis.

It should be noted that this process of confirming one hypothesis by matching the hypothesis and then rejecting other, competing hypotheses is one which is generally interwoven throughout the process of clinical cognition. For example in the present illness, the working hypothesis might concern the 'facts' concerning some piece of the history, with competing hypotheses providing alternative interpretations of what really happened to the patient at the time in question. The same issues of confirmation, rejection, and weighing likelihoods are relevant here even though the hypotheses are not about diseases, but rather about the facts themselves.

3) Alteration

It was noted above that the initial context chosen by the clinician is often not supported by the information subsequently gathered. Hence the context must be replaced by a new one. If the clinician is to operate effectively and efficiently in the clinical environment, he must generally be able to shift smoothly from one hypothesis to another. The process by which this replacement occurs is an important and interesting one.

One hypothesis is that the facts are again sifted through the pattern matching processes mentioned above, and from this re-examination of the data, a new hypothesis emerges as the working context. There seems little doubt that this happens in some situations, but as a general rule, such a process seems more characteristic of a medical student or a new intern than of an experienced clinician. For the latter, a more much directed move to a new hypothesis seems appropriate. That is the expert, because of his richer and more extensive experience uses certain 'failures' in matching findings to hypotheses as direct pointers to new hypotheses. Thus, for example, the working context might be 'glomerulitis', and a questionable fit of the facts has been found; the patient has heavy proteinuria but no significant hematuria. The expert responds to this 'mismatch' by moving directly to the 'nephrotic syndrome' context, because he has been in this situation a sufficient number of times to have stored the 'contingency' pointer.

The importance of these direct 'pointers' arises from the amount of structure which they preserve. In general, a reasonable amount of cognitive effort has gone into the 'fleshing-out' of the working hypothesis, and a lot of information has been gathered. If the hypothesis is simply abandoned, and no other one is directly taken up in its place, the clinician may lose track of certain pieces of information. If the new hypothesis can be obtained directly from the old one, then this smoother transition is apt to disrupt less severely the information structure he has built.

4) Dealing with Novelty

What does the clinician do when none of his working hypotheses seems consistent with the facts at hand? Such a situation can easily occur. For example, the might be one or more facts which are in error. Alternatively, the patient might be suffering from more that one disease, and the findings cannot all be attributed to one of them.

Because such situations clearly arise in clinical practice, the good clinician will have developed strategies for dealing with them. We do not know much about these strategies at present, but we will offer a few observations. First, there is always the possibility that the clinician is facing a situation which is truly novel in certain very important regards. In this case, he will have to fall back on general intelligence and 'creativity', but we cannot offer much detail about how this is done. Undoubtedly he begins his search for an understanding of the situation by trying to understand what modifications of contexts which 'almost fit' would be required. From these necessary modifications he may be able to move to a better grasp of the situation.

In other cases, the working hypothesis seems basically sound, but certain facts cannot be fitted into the framework it provides. At face value the situation may appear novel, but the clinician suspects that either one or more 'facts' are in error, or there is some alternative 'explanation' of the facts which will fit into the current context. This situation is discussed in more detail in a later section which considers how clinicians deal with discrepant information.

5) Learning

The abilities described above are in some sense a minimal set for an expert to have if he is to perform as an expert. We know that he possesses cognitive mechanisms to realize these abilities because we can observe him successfully dealing with the problems of clinical medicine, and this task environment requires these skills.

Because experts are not created de novo, however, they must possess the skills required to become experts. They must possess the ability to learn. In terms of our above discussion, they must be able to assimilate new contexts, recognition patterns, explanations of discrepancies, and administrative strategies. This assimilation draws from a variety of sources: school, books, clinical experience, introspection, etc. Further, it is clear that simple assimilation is not sufficient for expert behavior. The knowledge that is assimilated must be organized by the learner so that it is effectively available to him in the task environment of clinical practice.

The question of whether a piece of information has been effectively assimilated into the knowledge structure possessed by the clinician can be judged only with respect to the way in which the new knowledge is used in the above processes. Hence it seems that a prerequisite for understanding learning as it relates to clinical expertise is the understanding of <u>performance</u> in the clinical environment.

## The Initial Theory

Our theory of the cognitive behavior of clinicians is an amalgam of the ideas of a number of the workers in our group and was strongly influenced by Minsky [13]. Particularly notable contributions to the structure of this theory were made by Sussman, Pauker, and Rubin. Although our current theory is primitive and incomplete, we believe that it represents a good beginning. Here we will present it in some detail. Basically this presentation is a re-working of the above discussion in terms of the computer-based model we have implemented. The concepts used in that model are introduced at appropriate points in the discussion.

## Frames

It seems that the knowledge possessed by a clinician is grouped into chunks, which, after Minsky [ref], we call <u>frames</u>. When he begins to entertain a certain diagnostic possibility, be it a disease, like acute post-streptococcal glomerulonephritis, a clinical state, like nephrotic syndrome, or a physiological state, like sodium retention, he bring many facts about this possibility to mind at once. It appears that physicians behave as if certain findings, which he have called <u>triggers</u> serve to awaken the frame into our consideration. (This is the basic mechanism for dealing with the problem of <u>expectation</u> discussed earlier.) At that point, any of its findings or <u>slots</u> can relate to presented data, but when it was in its dormant state, most of these slots could not react to presented data. For example, when told of fever, one would not immediately think of cellulitis (a kind of skin infection), but if told that there was a red, painful swelling of one cheek, the additional finding of fever fits in neatly.

Frames appear to have other types of data associated with them besides slots. There appear to be relational pointers to other frames, so that when one is considering one frame as a possibility, one is "sort of" thinking about other related frames. This relationship may be of several varieties, but a neat grouping of many of them can be made by considering the <u>causes-of</u>, things <u>caused-by</u>, <u>complications-of</u>, and things <u>complicated-by</u> the frame. For example, when one is considering acute glomerulonephritis, one "sort of" thinks about acute renal failure and acute hypertension, both of which are complications of AGN, but they are not thought of in the same detail as AGN, e.g., one usually does not consider their complications, like encephalopathy, hyperkalemia,etc, unless other data suggests them or reinforces the hypotheses of acute renal failure and acute hypertension.

## Differential Pointers

In addition, there appear to be some special kinds of slots which function as lateral or differential pointers to other frames. These are meant to handle unexpected finding in a fashion that makes backing-up (a relatively costly procedure) less necessary. Rather than going back to the beginning and 'reshuffling' all the facts when a hypothesis is rejected, it appears that the physician has certain heuristics which point in specific directions when certain inconsistencies are encountered. This is a part of their response to the problem of alteration discussed earlier. For example, when presented with a patient with massive edema and heavy proteinuria, the expert can leap to a hypothesis of nephrotic syndrome. If he later discovers the patient has jugular venous distension, he can move directly to considering constrictive pericarditis, realizing that the two entities can be confused. This lateral motion is not based on reconsideration of all the data at hand, but on the differential pointer that says:

> "If you are considering nephrotic syndrome, and there is neck vein distension, then consider constrictive pericarditis."

Similarly, a young man with facial edema and hypertension can be hypothesized to have acute glomerulonephritis, since the unexpected findings of hypertensive retinopathy or ventricular hypertrophy on electrocardiogram would immediately lead to consideration of chronic glomerulonephritis.

## Pruning Frames

It also seems that the physician does not maintain multiple copies of diseases having certain variations, but rather he has a general knowledge and certain rules about how to tailor-make this to the case at hand. We call this process pruning. Pruning is related to the problems of elaboration and alteration discussed earlier. Pruning may involve findings (slots), evaluations or relationships to other frames. Thus, the general picture of cirrhosis must be modified in that one cannot expect to consider gynecomastia in a women. Sodium retention may be manifested by pedal edema, facial edema, ascites and the like, but ascites is rare in renal edema and facial edema is rare in cardiac edema, even though both are part of the physician's general knowledge about sodium retention. Sodium retention may be caused by cirrhosis in the adult, but rarely in children, so when considering sodium retention one should not "sort of" consider cirrhosis, if it is a child.

## Translation Frames

Another type of knowledge which physicians often bring to bear on their diagnoses relate not so much to the specific disease entities, but to a general knowledge about the world in general and medicine in particular.

Much of this knowledge can be expressed in a special kind of frame which we have called a translation frame. In some ways this can be viewed as a simple stimulus-response set:

> "If one is told the patient served in the army, it means he most likely did not have hypertension or proteinuria at that time (he passed an army physical), he probably did not have a murmur (army physicals are not known for careful observation), and probably had reasonable exercise tolerance."

> "If the patient attended summer camp, he was likely exposed to plant allergens,snake bite, other children and therefore common childhood diseases of summer (like the enteroviruses)."

## Hypothesis Generation

There appears to be a hierarchy of hypothesis in so far as how actively they are being considered and in comparing them to each other. There appears to be several general classes of consideration which he have called happy, active, semi-active, and dormant.

When beginning consideration of any problem, all hypotheses are dormant; that is to say, only their trigger slots can grasp incoming data. Under specified conditions, usually finding a datum to satisfy a trigger slot, the frame moves into active state. This means that any of its slots can match findings (with the constraint that they may be pruned in fitting the frame to the case at hand). The neighbors (e.g., causes-of, complications-of, etc.) of the frame are "sort of" made active. We call their level of activity semi-active. It differs from full activity in that its "awakening" does not awaken its neighbors, thus avoiding the explosive awakening of too many frames. Finally, under certain conditions, frames become happy, that is to say, they are convinced beyond reasonable doubt that they are true and they assert that they are indeed true so that other conclusions may proceed from this assertion.

## Hypothesis Testing

As findings are gathered, each frame is evaluated in several ways:

1) A check is made to see if the new datum excludes that frame. For example, the absence of proteinuria virtually denies the existence of a glomerulitis.

2) A check is made whether data is sufficient to establish the hypothesis. For example, if one finds red cell casts in the urine sediment, this virtually establishes the presence of a glomerulitis.

3) A measurement is made of how well the data fit the hypothesis and how much of the data are explained by the hypothesis. These are two complementary measures and the clinician considers some combination of

them. If the goodness of fit exceeds a certain level, he might say that the "weight of evidence" would allow the frame to become happy. On the other hand, if the fit is sufficiently poor, one might drop the hypothesis from active consideration. In doing this <u>scoring</u>, the physician allows for propagation through relations, i.e., if one is considering aortic stenosis and congestive heart failure, the finding of rales in the chest examination is very helpful to the congestive heart failure hypothesis, but by helping that hypothesis, it "sort of" lends weight to aortic stenosis also.

This then represents the substrate of the initial theory of the response of the clinician to the presentation of information about the patient. The theory has certain additional features which we can call <u>heuristic</u> rules, or what to do in certain situations. An example might be how to handle contradictory data:

> If one is told there are both red blood cell casts on urine sediment and no hematuria, then consider that there are probably no red cell casts (they are often confused with other casts) , but at some later time, see how your conclusions would be altered if red cell casts were present.
>
> If renal function is normal but you are told that there are no kidneys on x-ray of abdomen, consider the possibility that there are really large kidneys present, but the radiologist did not see them (as often happens with really large kidneys).

<u>Information Seeking</u>

At present, our theory of how the clinician chooses what facts to seek out is somewhat underdeveloped. We do have some understanding of this process, however, and this is a problem which is currently under study.

First it is clear that what may appear to be a "fact" to an outside observer may be less than that to a clinician. By this we mean that clinicians seem to deal in "chunks" of information which are, strictly speaking, composed of more than one fact. For example, a clinician tends to follow rather set patterns of questions until he has gotten a chunk of information about the patient. If the complaint is edema, a renal specialist will react by invoking a small "subroutine" to further characterize the edema. We call this a subroutine because clinicians themselves seem to recognize the questioning net they use as an automatic response to the stimulus "edema".

The rationale for the particular sequence of questions employed is understood by the physician, and he can readily explain it. But in practice, he does not "derive" this sequence, but rather simply remembers and invokes it.

Once a suitable chunk of information has been gained, the triggering and matching processes described above are invoked.

For the selection of which chunk of information to seek next, the clinician appears to make use of the frames themselves, trying to fill in the slots of his current hypothesis. Our understanding of the details of this process is inadequate at present, but we have been able to get some interesting results in our computer simulation by following this simple strategy.

The following few sections discuss specific projects which we have undertaken in support of the development of this theory. The first is the computer simulation of the present illness. The second project is concerned with style differences among clinicians insofar as their approach to the present illness is concerned and with measuring the effectiveness and efficiency which these differences promote. The third project is concerned with the development of orderly and concise means for identifying and codifying clinical knowledge, particularly of the kind found in medical textbooks. This work is aimed at filling some of the gaps which the present illness project must necessarily leave as it concentrates on strategy.

<u>Initial Computer Simulation of Cognitive Process</u>

In conjunction with our explorations of the knowledge and problem-solving behavior of clinicians described in the preceding sections, we have developed some preliminary computer programs to simulate aspects of the observed process of taking a present illness.

We will provide only some of the details of the operations of the computer programs involved to give the reader the flavor of our work. It should be understood, however, that these details will almost certainly be changed. In fact, much of the work discussed below in the section on supporting computer science research is aimed at refining and improving the mechanisms upon which this rudimentary simulation is built.

The basic operation of the simulation program is as follows. The age and sex of the patient is presented to the program along with the chief complaint. The program responds to this information by formulating hypotheses about the patient's condition. These hypotheses are the result of patterns of signs and symptoms which the program recognizes as suggestive of particular diseases, clinical states, or pathophysiological states. For example, the pattern "middle-aged man with pedal edema" might suggest idiopathic nephrotic syndrome, sodium retention, etc. The pattern currently known to the program were identified in our studies of experts, and the program makes the same use of them that the experts do, namely to immediately get one or more working hypotheses around which it can structure the initial phases of the present illness.

In the current simulation, the program must seek out all additional information about the patient. Therefore, once it has "digested" the age and sex and presenting complaint of the patient, it undertakes questioning of the user to learn more about the patient. Whenever a new fact is learned, the program revises its assessment of various hypotheses, and then seeks more information in accordance with its latest "opinion" of the situation. To understand the simulation, then, we need to understand two basic functions of the program:

1) how hypotheses are generated and tested
2) how questions are selected.

Here we will briefly investigate each of these questions. As noted, the emphasis will be on the concepts involved, not on the technical details of the program.

### Hypothesis Generation

Stored in a data base used by the program are a great many patterns of signs and symptoms. Associated with each pattern is some action which the program is to take if the pattern is found during the present illness. Some of the actions affect hypotheses, in that they cause hypotheses to be formed, modified, or deleted. Other types of patterns and their uses will be discussed below.

The patterns of findings which cause hypotheses to be promoted to active consideration are called *triggers*. At the beginning of the present illness, all hypotheses are *dormant* in that although the program has descriptive knowledge about them (See the discussion of frames below.), it is not *actively* considering any of them. The triggers are used to promote some hypotheses to the *active* state when the chief complaint is entered. (Triggers are used at other points in the present illness also, as we shall see.) While a hypothesis is active, the program matches new facts to the description of the hypothesis (the frame) which is has been given, and it uses the frame for the hypothesis in its question selection activities. On the other hand, dormant hypotheses are ignored in both these activities.

So a trigger moves a hypothesis from the dormant state to the active state. In doing so, it may cause other hypotheses to move from the dormant state to a state which we have called *semi-active*. To understand the purpose of this third state, consider the above example, namely the presenting problem of massive pedal edema in a middle-aged man. There are triggers which cause the hypotheses of nephrotic syndrome, idiopathic nephrotic syndrome, and sodium retention (among other things) to become active. To reflect the fact that at this point a clinician would "sort of" be thinking of congestive heart failure (because it is a cause of sodium retention), the program moves congestive heart failure to the semi-active. The simulation program matches findings to semi-active hypotheses, but it does not use them in its question selection activities.

The specific rule which the program uses to determine which hypotheses to move into the semi-active state when a trigger is matched is as follows. The program looks at the description (frame) for the hypothesis denoted by the trigger, and finds all hypotheses related to the hypothesis in question by such relations as "causes", "complication-of", etc., and makes these hypotheses semi-active (assuming, of course, that they are not already active).

Hypotheses can move from the semi-active state to either the active state or to the dormant state as the present illness proceeds. For example, if a later finding is a trigger for a semi-active hypothesis, the latter will move to the active state. In addition, a hypothesis can move from semi-active to active if more than one other hypothesis, in becoming active, tries to move the hypothesis in question to semi-active status.

In fact, throughout the present illness, there is continual movement of hypotheses from one state to another. Active hypotheses may be "demoted" to dormant by the hypothesis testing function because it deems them to be very poor fits to the facts. The important point, however, is that hypotheses are being re-evaluated and re-ranked by the program in light of the most recent set of facts about the patient.

Consider Figure 1. Here is the trace of the simulation program as it responds to the presentation of massive pedal edema in a middle-aged man. The age and sex descriptor are translated into internal format, where each property is labeled by type. When massive pedal edema is entered, we see that this <u>triggered</u> sodium retention and nephrotic syndrome, which in turn, cause their "relatives" (for example, congestive heart failure and acute tubular necrosis are causes-of sodium retention) to go into the <u>semi-active</u> state. When <u>idiopathic</u> nephrotic syndrome became semi-active, it discovered that a prior fact (the age descriptor) fitted neatly into its description, and this second match allowed the frame (<u>idiopathic</u> nephrotic syndrome) to rise to full activity). This did not occur when the age descriptor was intially given because that finding was not a trigger for the frame. The frame had to be at least semi-active (rather than dormant) before the match could occur.

Similar interactions occur with chronic renal failure and chronic glomerulonephritis, but the reason that they come to full activity is not that they find a supporting finding, but rather that they are "sort of" thought about by more than one other frame (in this case, sodium retention and nephrotic syndrome).

In Figure 2 is a tabulation of the state of the hypotheses considered by the program. It is easy to see how this might be transformed into a "problem list" with relatively little effort. Each frame has two associated measures: its score is a normalized measure (from -1 to 1) of how well the data fits the frame, and is EXPL is the fraction of findings explained by the frame and its possible associated subframes.

[redacted]

```
==========================================================================

>  (MIDDLE-AGED MAN)

>>>>>>> (MAN (AGE MIDDLE-AGED) (TIME NOW))

>  (MASSIVE PEDAL EDEMA)

>>>>>>> (EDEMA (LOCATION PEDAL) (SEVERITY MASSIVE) (TIME NOW))

((EDEMA (LOCATION PEDAL) (SEVERITY MASSIVE) (TIME NOW))
 ==TRIGGER==>
 ((SODIUM RETENTION)<-- ACTIVE
  ==>
  ((CONGESTIVE HEART FAILURE) <-- SEMI-ACTIVE
   (CIRRHOSIS) <-- SEMI-ACTIVE
   (ACUTE TUBULAR NECROSIS) <-- SEMI-ACTIVE
   (NEPHROTIC SYNDROME) <-- SEMI-ACTIVE
   (ACUTE GLOMERULONEPHRITIS) <-- SEMI-ACTIVE
    ==>
    ((NEPHROTIC SYNDROME) <-- ACTIVE
      ==>
     ((DIABETES) <-- SEMI-ACTIVE
      (SYSTEMIC LUPUS) <-- SEMI-ACTIVE
      (IDIOPATHIC NEPHROTIC SYNDROME) <-- SEMI-ACTIVE))))))

((AGE (AGE MIDDLE-AGED) (TIME NOW))
 ==TRIGGER==>
 ((IDIOPATHIC NEPHROTIC SYNDROME) <-- ACTIVE))

((EDEMA (LOCATION PEDAL) (SEVERITY MASSIVE) (TIME NOW))
 ==>
 ((NEPHROTIC SYNDROME)
   -->
   ((INSECT BITE) <-- SEMI-ACTIVE
    (NEPHROTOXIC DRUGS) <-- SEMI-ACTIVE
    (CHRONIC GLOMERULONEPHRITIS) <-- SEMI-ACTIVE
    (GLOMERULITIS) <-- SEMI-ACTIVE
    (CELLULITIS) <-- SEMI-ACTIVE
    (HYPOVOLEMIA) <-- SEMI-ACTIVE
    (CHRONIC GLOMERULONEPHRITIS) <-- ACTIVE
     ==>
     ((CHRONIC RENAL FAILURE) <-- SEMI-ACTIVE)))

 ==>
 ((CHRONIC RENAL FAILURE) <-- ACTIVE
  ==>
  ((UREMIA) <-- SEMI-ACTIVE
   (HYPERKALEMIA) <-- SEMI-ACTIVE))
 ==>
 (CHRONIC GLOMERULONEPHRITIS) --> ((CHRONIC HYPERTENSION) <-- SEMI-ACTIVE
                                  (FOCAL GLOMERULONEPHRITIS) <-- ACTIVE))

==========================================================================
```

**FIGURE 1. HYPOTHESIS GENERATION**

(NOTE: User input preceded by single '>'.)

[redacted]



```
=========================================================================

 (MAN (AGE MIDDLE-AGED) (TIME NOW))
 (SEX (GENDER MALE) (TIME NOW))
 (AGE (AGE MIDDLE-AGED) (TIME NOW))
 (EDEMA (LOCATION PEDAL) (SEVERITY MASSIVE) (TIME NOW))
 (BOUND (EDEMA (LOCATION PEDAL) (SEVERITY MASSIVE) (TIME NOW))
 (SODIUM RETENTION)
 (EDEMA SODIUM RETENTION))
 ((SODIUM RETENTION) ACTIVE)
 (PRUNED-SLOTS (SODIUM RETENTION) ((DIURETIC SODIUM RETENTION)))

HAPPY-FRAMES
  NONE

ACTIVE-FRAMES

   (IDIOPATHIC NEPHROTIC SYNDROME) SCORE 0.165 EXPL 0.5 AVG 0.332
   (NEPHROTIC SYNDROME) SCORE 0.151 EXPL 0.5 AVG 0.325
   (SODIUM RETENTION) SCORE 0.102 EXPL 0.5 AVG 0.301
   (CHRONIC RENAL FAILURE) SCORE 0.071 EXPL 0.5 AVG 0.285
   (FOCAL GLOMERULONEPHRITIS)
   (CHRONIC GLOMERULONEPHRITIS)

SEMI-ACTIVE-FRAMES

   (ACUTE GLOMERULONEPHRITIS) SCORE 0.097 EXPL 0.0 AVG 0.048
   (CHRONIC HYPERTENSION)
   (HYPERKALEMIA)
   (UREMIA)
   (HYPOVOLEMIA)
   (CELLULITIS)
   (GLOMERULITIS)
   (NEPHROTOXIC DRUGS)
   (INSECT BITE)
   (SYSTEMIC LUPUS)
   (DIABETES)
   (ACUTE TUBULAR NECROSIS)
   (CIRRHOSIS)
   (CONGESTIVE HEART FAILURE)

=========================================================================
```

FIGURE 2. FACTS AND HYPOTHESES

The details of the scoring scheme are discussed below in connection with hypothesis testing.

## Hypothesis Matching

In the above discussion, we ignored the representation of knowledge about diseases, clinical states, etc. used by the simulation program. We did not need this detail in our discussion of the triggering mechanism and the various states for for hypotheses.

One of the major activities of the present illness simulation program, however, is assessing how well the facts in hand at any point in time match a given hypothesis. Therefore, we need to examine the way in which descriptions of hypotheses are stored and used.

Each description is represented by a frame. A frame is an organized collection of facts about the hypothesis, what its findings are, how it is caused, what complications can arise from it, etc.

Because medical knowledge generally is organized about diseases or clinical states, and not about the implications of specific findings, this system allows for data input as its is available in standard medical texts. The necessary cross referencing for the appropriately useful associations is taken care of automatically by a frame compiler. Figure 3 is an example of a typical frame. This frame might be paraphrased as:

> Nephrotic syndrome is a clinical state characterized by hypoalbuminemia, heavy proteinuria (usually over 5 grams in a 24-hour urine), massive edema, symmetrically distributed, often involving the face, especially ther area about the eyes. There is associated elevation of serum cholesterol and urine lipids are present. It may be caused by acute or chronic glomerulonephritis, nephrotoxic drugs, some insect bites, diabetes, systemic lupus, diabetes, or may be idiopathic. It may be complicated by hypovolemia (intravascular) or infection of the massively swollen extremities. There is almost never facial edema in the absence of pedal edema, and massive edema associated with over 5 grams of protein loss daily is enough to establish the diagnosis. It may be confused with constrictive pericarditis, but in that case there is neck vein elevation. It may also be confused with cirrhosis, but in that case, ascites are usually present. If there is flank pain, one must consider renal vein thrombosis as a possible cause of the renal protein loss.

Now we can explore the scoring or hypothesis matching performed by the simulation program. Consider the scoring data shown in Figure 3, under the titles MAJOR and MINOR.

```
(DEFRAME
 $(NEPHROTIC SYNDROME)
 (TYPE CLINICAL-STATE)
 (SLOT ALB (TRIGGER) $(ALBUMIN LOW))
 (SLOT PRO NIL $(PROTEINURIA HEAVY))
 (SLOT PROQ (TRIGGER) $(PROTEINURIA >5GRAMS))
 (SLOT EDEMA (TRIGGER) $(EDEMA MASSIVE (NOT ASYMMETRICAL)))
 (SLOT FACED (TRIGGER) $(EDEMA (OR FACIAL PERI-ORBITAL) (NOT ASYMMETRICAL)))
 (SLOT CHOL NIL $(CHOLESTEROL HIGH))
 (SLOT URFAT NIL $((URINE LIPIDS) PRESENT))
 (CAUSED-BY $(ACUTE GLOMERULONEPHRITIS)
      $(CHRONIC GLOMERULONEPHRITIS)
      $(NEPHROTOXIC DRUGS)
      $(INSECT BITE)
      $(IDIOPATHIC NEPHROTIC SYNDROME)
      $(SYSTEMIC LUPUS)
      $(DIABETES))
 (COMPLICATED-BY $(HYPOVOLEMIA) $(CELLULITIS))
 (MAJOR #((SS(ALBUMIN LOW) 1.0)
   (SS(ALBUMIN HIGH) -1.0))
 #((SS(PROTEINURIA >5GRAMS) 1.0)
   (SS(PROTEINURIA HEAVY) 0.5)
   (SS(PROTEINURIA (OR ABSENT LIGHT)) -1.0))
 #((SS(EDEMA MASSIVE (NOT ASYMMETRICAL)) 1.0)
  (SS(EDEMA (NOT ABSENT) (NOT ASYMMETRICAL) (NOT ASYMMETRICAL)) 0.3)
   (SS(EDEMA ERYTHEMATOUS (NOT ABSENT)) -0.2)
   (SS(EDEMA ABSENT) -1.0)))
 (MINOR #((SS(CHOLESTEROL HIGH) 1.0)
   (SS(CHOLESTEROL (NOT HIGH)) -1.0))
 #((SS((URINE LIPIDS) PRESENT) 1.0)
   (SS((URINE LIPIDS) ABSENT) -0.5)))
 (MUST-NOT-HAVE $*(AND (EDEMA FACIAL (NOT ABSENT)) (EDEMA PEDAL ABSENT)))
 (IS-SUFFICIENT $* (AND (EDEMA MASSIVE) (PROTEINURIA >5GRAMS)))
 (DIFFERENTIAL-DIAGNOSIS
  ($((NECK VEINS) ELEVATED)
   (SEMI-ACTIVATE '$(CONSTRICTIVE PERICARDITIS)))
  ($(ASCITES PRESENT) (SEMI-ACTIVATE '$(CIRRHOSIS)))
  ($(FLANK-PAIN)
   (SEMI-ACTIVATE '$(RENAL VEIN THROMBOSIS)))))
```

**FIGURE 3. NEPHROTIC SYNDROME FRAME**

The score information given in each frame consists of a list of various tests, associated with a number between -1 and 1. If the test is true, that number is added to an accumulating sum. The maximum sum is the total number of such items, so a normalized score is the actual sum divided by the maximum. If no data is known about the fact sought, zero is added to the actual sum, so this weighs somewhat against the score, but less so as more data is known since the sum is divided by a larger normalizing factor. Major and Minor scores just specify factors by which there respective sums are multiplied, so the major factors count more. Score propagation is accomplished by passing the score of the related frame (not its sum), which is therefore normalized already, as an additional test. Frames may move from one state to another (e.g., from active to semi-active) when certain logical criteria are met. (A positive throat culture is sufficient to establish a streptococcal infection), but we also allow changes based on weight of evidence. For example, is the score of any active frame exceeds a pre-established threshold, then it becomes happy, whereas if it falls below a different pre-established threshold, it may lapse into the semi-active state.

At this point we might digress to mention score-propagation . It is clear that when a frame gains evidence in its behalf, its relatives must also become more convinced of their truth also. For example, acute glomeronephritis is related to (by "complicated-by") acute hypertension. If we learn, that there is hypertension in the absence of hypertrophy on the electrocardiogram, this must add weight to acute glomerulonephritis. If we then learn that there is no chronic hypertensive retinopathy, acute hypertension gains more credence, and this gain must be propagated up to acute glomerulonephritis.

The inverse effect is equally true, i.e., since a low urine sodium is explained by sodium retention, and since sodium retention can be caused-by acute glomerulonephritis, then acute glomerulonephritis can explain the abnormal finding of low urine sodium if we can invoke sodium retention. In this program, both scores and "explanations" of findings can be propagated through frames which are either happy or active.

## Question Selection in the Present Illness

Now we can turn our attention to the way in which the program seeks additional information during the present illness. Here we have implemented procedures which are first approximations to those the program will need if it is to behave in the style of a physician in so far as its choice of and ordering of questions is concerned

From our detailed study of the way in which a particular expert took a present illness, we concluded that he used two distinct modes of questioning. At times, he invoked a rather rigid, "compiled", sequence of questions, particularly to sharpen the characterization of a particular finding. This sequence seemed aimed at quickly, but narrowly, focusing the problem solving. Such questions can be thought of as filling a pattern which if matched will trigger a very specific hypothesis. An example of such a sequence is shown in the first part of Figure 4.

The program is first told that the patient is a young boy with facial edema (at this point, it might be well to say that the patient who is being questioned in this example actually has acute glomerulonephritis). The program attempts to further characterize the facial edema, asking about duration, recurrence, temporal pattern, etc. The edema fits so well into the typical pattern of renal edema, that the program does not pursue details such as pain and erythema. At this point, the chances that this is anything other than renal edema are so remote that the program is willing to pay a "reprocessing penalty" if it is wrong.

Next the program asks about associated pedal edema. This occurs because of a simple heuristic rule which states: "if you are told of facial edema, see if there is associated pedal edema ." (This fits with the MUST-NOT-HAVE rule in the Nephrotic Syndrome frame <Figure 3>). Pedal edema is likewise explored in depth, but note the additional questions about severity, pain and erythema which are relevant for this kind of edema. It should be noted that the determination of what is relevant here is the behavior of the expert himself. He asks different questions about pedal edema than about facial edema, and in order for the program to appear to behave "naturally", it must do the same.

This additional question can also be thought of as being more or less "automatic" in that the common conjunction of pedal and facial edema makes the investigation of the former a natural next step for the clinician, and hence for the program.

It is at this point that the second form of questioning makes its appearance. Here the clinician must weigh various hypotheses and possible questions and seek to plot a good strategy.

We have suh()essed the detailed output of the program as it does the hypothesis generation and matching described above. Suffice it to say that at this point, the program examines the states and scores of the various frames in deciding on its next question. It will pursue the active frame (and its related subframes) which has the highest score. It will not pursue happy frames, since it is already reasonably convinced of their applicability.

The score used in selecting the frame to explore is not just the "goodness of fit" score mentioned above. Since the choice is guiding further questioning, we are also concerned that our hypothesis (and its subframes) explain as much as possible of the data. Hence the program averages the SCORE and EXPL values, and pursues the hypothesis with the highest average. In this case, that is acute glomerulonephritis, and the program turns to one of the latter's subframes , streptococcal infection. When it finds that there is indeed a pharyngitis, it explores that in depth as to character and severity.

```
==================[redacted]======================================
>> (YOUNG BOY)
>> (FACIAL EDEMA)
ABOUT (NOW FACIAL EDEMA),
CONCERNING DURATION,IS IT:
1.    DAYS
2.    WEEKS
3.    MONTHS
4.    YEARS
>> 1
CONCERNING RECURRENCE,IS IT:
1.    FIRST-TIME
2.    INFREQUENT
3.    OCCASSIONAL
4.    FREQUENT
>>
CONCERNING PERI-ORBITAL?,IS IT:
1.    PERI-ORBITAL
2.    NOT-PERI-ORBITAL
>> 1
CONCERNING SYMMETRY,IS IT:
1.    SYMMETRICAL
2.    ASYMMETRICAL
>> 1
CONCERNING TEMPORAL-PATTERN,IS IT:
1.    NO-TEMPORAL-PATTERN
2.    WORSE-IN-MORNING
3.    WORSE-IN-EVENING
>> 2

IN THINKING
ABOUT (PEDAL EDEMA),
CONCERNING TIME,IS IT:
1.    NOW
2.    RECENT-PAST
3.    PAST
>> 1
CONCERNING STATUS,IS IT:
1.    ABSENT
2.    PRESENT
>> 2
CONCERNING DURATION,IS IT:
1.    DAYS
2.    WEEKS
3.    MONTHS
4.    YEARS
>> 1
CONCERNING RECURRENCE,IS IT:
1.    FIRST-TIME
2.    INFREQUENT
3.    OCCASSIONAL
4.    FREQUENT
>> 1
CONCERNING TEMPORAL-PATTERN,IS IT:
1.    NO-TEMPORAL-PATTERN
2.    WORSE-IN-MORNING
3.    WORSE-IN-EVENING
>> 3
=====================================================================
```

FIGURE 4. PRESENT ILLNESS SIMULATION

(Note: user inputs preceded by '>>')

```
************************************************************************
CONCERNING SEVERITY,IS IT:
1.    1+
2.    2+
3.    3+
4.    4+
5.    MASSIVE
>> 3
CONCERNING SYMMETRY,IS IT:
1.    SYMMETRICAL
2.    ASYMMETRICAL
>> 1
CONCERNING TYPE,IS IT:
1.    PITTING
2.    NON-PITTING
>> 1
CONCERNING ERYTHEMA,IS IT:
1.    ERYTHEMATOUS
2.    NOT-ERYTHEMATOUS
>> 2
CONCERNING PAIN,IS IT:
1.    PAINFUL
2.    NOT-PAINFUL
>> 2

PLEASE TELL ME ABOUT
 PHARYNGITIS,I.E.,
 IS THERE (NOW (NOT ABSENT) PHARYNGITIS) ?
>> YES
ABOUT (NOW (NOT ABSENT) PHARYNGITIS),
CONCERNING APPEARANCE,IS IT:
1.    EXUDATIVE
2.    NON-EXUDATIVE
>> 2
CONCERNING SEVERITY,IS IT:
1.    MILD
2.    SEVERE
>> 2

HAPPY-FRAMES
    NONE

ACTIVE-FRAMES
   (ACUTE GLOMERULONEPHRITIS) SCORE 0.298 EXPL 0.75 AVG 0.524
   (SODIUM RETENTION) SCORE 0.195 EXPL 0.75 AVG 0.472
   (STREPTOCOCCAL INFECTION) SCORE 0.181 EXPL 0.75 AVG 0.465
   (IDEOPATHIC NEPHROTIC SYNDROME) SCORE 0.161 EXPL 0.75 AVG 0.455
   (NEPHROTIC SYNDROME) SCORE 0.068 EXPL 0.75 AVG 0.409
   (ACUTE RENAL FAILURE) SCORE 0.066 EXPL 0.75 AVG 0.408

PLEASE TELL ME ABOUT
 STREPTOCOCCI,I.E.,
 IS THERE (NOW EXPOSURE STREPTOCOCCI) ?
>> ?

************************************************************************
```

FIGURE 4. Continued

```
PLEASE TELL ME ABOUT
 SCHOOL,I.E.,
 IS THERE (NOW ATTENDED SCHOOL) ?
>> YES

PLEASE TELL ME ABOUT
 PENICILLIN,I.E.,
 IS THERE (NOW GIVEN PENICILLIN) ?
>> ?

PLEASE TELL ME ABOUT
 FEVER,I.E.,
 IS THERE (NOW MILD FEVER) ?
>> NO

PLEASE TELL ME ABOUT
 (THROAT CULTURE),I.E.,
 IS THERE (NOW BETA (THROAT CULTURE)) ?
>> YES

PLEASE TELL ME ABOUT
 HEMATURIA,I.E.,
 IS THERE (NOW (NOT ABSENT) HEMATURIA) ?
>> YES
ABOUT (NOW (NOT ABSENT) HEMATURIA),
CONCERNING AMOUNT,IS IT:
1.    MICROSCOPIC
2.    GROSS
>> 1

PLEASE TELL ME ABOUT
 PROTEINURIA,I.E.,
 IS THERE (NOW (NOT ABSENT) PROTEINURIA) ?
>> YES
ABOUT (NOW (NOT ABSENT) PROTEINURIA),
CONCERNING AMOUNT,IS IT:
1.    LIGHT
2.    HEAVY
>> 1
CONCERNING QUAN-AMOUNT,IS IT:
1.    <100MGRAMS
2.    100MGRAMS-5GRAMS
3.    >5GRAMS
>> ?

PLEASE TELL ME ABOUT
 WEIGHT,I.E.,
 IS THERE (NOW (OR HIGH RISING) WEIGHT) ?
>> NO

PLEASE TELL ME ABOUT
 RALES,I.E.,
 IS THERE (NOW PRESENT RALES) ?
>> YES
```

FIGURE 4. Continued

[redacted]

```
*****************************************************************************

HAPPY-FRAMES
   (STREPTOCOCCAL INFECTION) SCORE 0.348 EXPL 0.538 AVG 0.443
ACTIVE-FRAMES
   (ACUTE GLOMERULONEPHRITIS) SCORE 0.477 EXPL 0.538 AVG 0.508
   (GLOMERULITIS) SCORE 0.287 EXPL 0.538 AVG 0.413
   (SODIUM RETENTION) SCORE 0.208 EXPL 0.538 AVG 0.373
   (IDEOPATHIC NEPHROTIC SYNDROME) SCORE 0.177 EXPL 0.538 AVG 0.358
   (CONGESTIVE HEART FAILURE) SCORE 0.110 EXPL 0.538 AVG 0.324
   (ACUTE RENAL FAILURE) SCORE 0.075 EXPL 0.538 AVG 0.307
   (ATHEROMATOUS EMBOLI) SCORE 0.005 EXPL 0.538 AVG 0.271
   (NEPHROTIC SYNDROME) SCORE -0.043 EXPL 0.538 AVG 0.247
   (STONE) SCORE 0.25 EXPL 0.076 AVG 0.163

  (NOW YOUNG BOY)


  (NOW FACIAL DAYS FIRST-TIME PERI-ORBITAL SYMMETRICAL WORSE-IN-MORNING EDEMA)
  (PEDAL NOW  PRESENT DAYS FIRST-TIME WORSE-IN-EVENING 3+ SYMMETRICAL PITTING
         NOT-ERYTHEMATOUS NOT-PAINFUL EDEMA)
  (NOW (NOT ABSENT) EXUDATIVE SEVERE PHARYNGITIS)
  ((STREPTOCOCCI (EXPOSURE EXPOSURE) (TIME NOW)) UNKNOWN)
  (NOW ATTENDED SCHOOL)
  ((PENICILLIN (GIVEN? GIVEN) (TIME NOW)) UNKNOWN)
  ((NOT MILD NOW) FEVER)
  (NOW BETA (THROAT CULTURE))
  (NOW (NOT ABSENT) MICROSCOPIC HEMATURIA)
  (NOW (NOT ABSENT) LIGHT PROTEINURIA)
  (NOW (NOT (OR HIGH RISING)) WEIGHT)
  (NOW PRESENT RALES)

*****************************************************************************
```

FIGURE 4. Continued

Next in Figure 4, we see the state of the various hypotheses that the program is considering.

Continuing its pursuit of streptococcal infection, the program looks for possible exposure. When it is told that no information about this is available, the program seeks indirect confirmation of the presumed exposure. he program finds that school attendance can result in streptococcal exposure. It makes this connection by tracking through a series of <u>translation</u> frames (streptococcal exposure goes along with childhood disease exposure, and the latter might occur in school or summer camp).

Finally the program is told that the throat culture was positive. With this fact, streptococcal infection becomes "happy", e.g. the program is , convinced that streptococcal infection is present, and asserts it as a finding. The program then turns to the next subframe of acute glomerulonephritis, since more data about streptococcal infection would at this point be moot. This next subframe is glomerulitis, and the issues of hematuria and proteinuria are pursued. At this point, sodium retention is explored.

In the bottom lines of Figure 4, we see a summary of the data in order of acquisition. To paraphrase:

> This is a young boy, who presents for the first time with symmetrical, peri-orbital edema, worse in the morning, for the past few days. It is associated, over the same time period, with 3+ symmetrical, pitting, pedal edema, which is worse in the evening. The patient has a severe, exudative pharyngitis, has been attending school, has not received penicillin and has not been febrile. Throat culture was positive for beta-hemolytic strep. There is also microscopic hematuria and light proteinuria. His weight is normal and has not been increasing, but rales are present on chest examination.

This is not an unreasonable first-pass present illness for a patient with acute glomerulonephritis.

A contrasting strategy is shown in Figure 5 where a different case is considered. After exploring the finding of pedal edema in a middle-aged man, a specific avenue of exploration is not clearly evident. Rather that beginning with in depth exploration of a particular frame, the program asks, in the manner of a clinician, about dyspnea, alcohol consumption, past proteinuria, varicose veins and jaundice, attempting to pin down an area for profitable exploration. Once the existence of past proteinuria has brought chronic glomerulonephritis, nephrotic syndrome and chronic renal failure to mind, these are then explored.

```
>> (MIDDLE-AGED MAN)
>> (SYMMETRICAL PEDAL EDEMA)
ABOUT (NOW SYMMETRICAL PEDAL EDEMA) ,
CONCERNING DURATION, IS IT:
1.    DAYS
2.    WEEKS
3.    MONTHS
4.    YEARS
>> 2
CONCERNING RECURRENCE, IS IT:
1.    FIRST-TIME
2.    INFREQUENT
3.    OCCASSIONAL
4.    FREQUENT
>> 3
CONCERNING TEMPORAL-PATTERN, IS IT:
1.    NO-TEMPORAL-PATTERN
2.    WORSE-IN-MORNING
3.    WORSE-IN-EVENING
>> 3
CONCERNING SEVERITY, IS IT:
1.    1+
2.    2+
3.    3+
4.    4+
5.    MASSIVE
>> 5
CONCERNING TYPE, IS IT:
1.    PITTING
2.    NON-PITTING
>> 1
CONCERNING ERYTHEMA, IS IT:
1.    ERYTHEMATOUS
2.    NOT-ERYTHEMATOUS
>> 2
CONCERNING PAIN, IS IT:
1.    PAINFUL
2.    NOT-PAINFUL
>> 2

IN THINKING
ABOUT (NOW DYSPNEA) ,
CONCERNING STATUS, IS IT:
1.    ABSENT
2.    PRESENT
>> 1

IN THINKING
ABOUT (NOW (ALCOHOL CONSUMPTION)) ,
CONCERNING AMOUNT, IS IT:
1.    NONE
2.    RARE
3.    SOCIAL
4.    HEAVY
5.    ALCOHOLIC
>> 3
```

FIGURE 5. ANOTHER PRESENT ILLNESS SIMULATION
(Note: user inputs preceedded by '>>')

```
=========================================================================

IN THINKING
ABOUT (PAST PROTEINURIA) ,
CONCERNING STATUS, IS IT:
1.    ABSENT
2.    PRESENT
>> 2
CONCERNING AMOUNT, IS IT:
1.    LIGHT
2.    HEAVY
>> 1
CONCERNING QUAN-AMOUNT, IS IT:
1.    <100MGRAMS
2.    100MGRAMS-5GRAMS
3.    >5GRAMS
>> ?

IN THINKING
ABOUT (NOW (VARICOSE VEINS)) ,
CONCERNING STATUS, IS IT:
1.    ABSENT
2.    PRESENT
>> 1

IN THINKING
ABOUT (NOW JAUNDICE) ,
CONCERNING STATUS, IS IT:
1.    ABSENT
2.    PRESENT
>> 1

HAPPY-FRAMES
     NONE
ACTIVE-FRAMES
  (CHRONIC GLOMERULONEPHRITIS) SCORE 0.213 EXPL 0.285 AVG 0.249
  (SODIUM RETENTION) SCORE 0.204 EXPL 0.285 AVG 0.245
  (NEPHROTIC SYNDROME) SCORE 0.166 EXPL 0.285 AVG 0.226
  (IDIOPATHIC NEPHROTIC SYNDROME) SCORE 0.166 EXPL 0.285 AVG 0.225
  (CHRONIC RENAL FAILURE) SCORE 0.086 EXPL 0.285 AVG 0.186
  (FOCAL GLOMERULONEPHRITIS)

 PLEASE TELL ME ABOUT
 KUB ,I.E.,
 IS THERE (NOW KIDNEYS-BOTH-SMALL KUB) ?
>> NO

PLEASE TELL ME ABOUT
 HEMATURIA ,I.E.,
 IS THERE ((NOT ABSENT) PAST HEMATURIA) ?
>> NO

PLEASE TELL ME ABOUT
 HYPERTENSION ,I.E.,
 IS THERE (NOW (NOT ABSENT) HYPERTENSION) ?
>> NO

=========================================================================
```

FIGURE 5. Continued

**Protocol Collection and Analysis**

Principals
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In conjunction with our studies of the clinical decision making process, we have undertaken the collection and analysis of tape-recorded protocols of a number of clinicians taking present illnesses. We really have two purposes in mind with respect to this study.

In the present illness project discussed above, we have relied on the observation of and introspection by a single clinical expert for the most part. Although this has proved very productive, we want to know if major variations in "style" exist, and whether some styles are more efficient and/or effective than others. Therefore, we need to broaden the base of the observed problem solving behavior upon which we are constructing our cognitive theory.

The second purpose of this study is to collect protocols which can be used in testing the computer simulations we are employing. With detailed protocols in hand, we can compare the behavior of programs with that of clinicians on a "step by step" basis. Such comparisons will undoubtedly suggest refinements and improvements in the theories, and this form of testing will be a central methodological tool of the Laboratory.

We have already initiated this collection and analysis of protocols. Our current study involves the presentation of a case to renal experts. The clinician is asked to take a present illness from the patient. (The part of the patient is played by another physician.) The basic procedure of the experiment is as follows:

1) The renal expert is first told the age, sex, and chief complaint of the patient.

2) The renal expert then can ask questions concerning the patient, one at a time.

3) For each question, he must say why he is asking the question.

4) After receiving the answer to a question, the expert must say what the answer "means" to him insofar as his current view of the case is concerned.

In the current study, we are presenting the same case to five renal experts on the staff of the New England Medical Center Hospital. This group was chosen for several reasons: 1) they are indeed experts, and we are interested in expert behavior; 2) they are kidney specialists, and their protocols on a kidney problem can be used in testing the simulation programs we are developing; and 3) because they are all in the same specialty and in the same hospital, they are apt to show some common behavior, and this will make our first pass at modeling their behavior somewhat easier.

As we become more experienced in the collection and analysis of protocols, and as our understanding of the clinical process becomes more highly developed, we will expand our efforts to include clinicians from other specialties.

As an example of a problem in which "style" differences might play an important role, consider the following.

Because the physician is often interested in historical information about the patient, he must often rely on the patient himself for this information. In many cases, the patient cannot (sometimes will not) remember the exact circumstances in question, or the recollections of the patient are suspect. In such a situation, the clinician may search for witnesses to the patient's past condition. Consider, for example this brief excerpt from a protocol in which the patient is a young boy with symptoms of heart disease and a possible episode of acute rheumatic fever some five years ago.

Pat. "Well, 4 or 5 years ago, I was out of school for 3 or 4 months. I had pain in my joints...."
Doc. "Tell me a little more about this episode. Were you hospitalized?"
Pat. "No. The doctor took care of me at home."
Doc. "What did he say was wrong with you?"
Pat. "St. Vitus dance."
Doc. "Did he treat you with anything?"
Pat. "He just gave me aspirin."
Doc. "He gave you aspirin? Did you take it frequently?"
Pat. "He said...you know... I don't even remember."
Doc. "Did you have a sore throat that started the whole thing off? Did anyone ever mention it to you? Did the doctor ask you whether you had a sore throat?"
Pat. "I don't know doc. I get a lot of sore throats."
Doc. "Did the doctor inject you with penicillin back in that time? Do you remember?"
Pat. "No he didn't inject me."
Doc. "You don't remember if you took any penicillin by mouth?"
Pat. "Oh, maybe he gave me some pills."
Doc. "Where's your mother?"

Now in this brief excerpt, we see the clinician trying to establish whether the patient in fact had an attack of acute rheumatic fever four or five years ago. The patient gives evidence which is not conclusive on the matter. The clinician turn his attention on the quest for